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ORIGINAL ARTICLE

Effects of Continuous Passive Motion on Reversing the Adapted Spinal Circuit in Humans With Chronic Spinal Cord Injury

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Abstract

Objective: To investigate the possibility of restoring the adapted spinal circuit after spinal cord injury (SCI) by means of long-term continuous passive motion (CPM) of the ankle joint.

Design: Randomized controlled trial with repeated measures.

Setting: Research laboratory in a general hospital.

Participants: Individuals with motor complete SCI (N = 14) were recruited from a community.

Intervention: CPM of the ankle joint for 1 hour a day, 5 days a week for 4 weeks.

Main Outcome Measures: Modified Ashworth Scale (MAS) scores for evaluation of spasticity and postactivation depression (PAD) were documented prior to and after intervention.

Results: MAS scores improved after 4 weeks of CPM intervention, indicating a reduction in spasticity of the ankle joint. PAD was restored after 4 weeks of training.

Conclusions: Passive motion of the ankle joint alone was sufficient in reversing the adapted spinal circuit, and therefore indicates that spasticity after SCI could possibly be managed by CPM intervention. The results of this study support the use of the passive mode of robot-assisted therapy for humans with complete SCI who cannot exercise actively.

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After injury to the spinal cord, the spinal circuitry undergoes various adaptations, which are paralleled by the progressive onset of the syndromes of spasticity.[1-3] Alterations of the spinal circuitry function, such as the reduction of postactivation depression (PAD) at the Ia-motoneuron synapse, could be one of the main spinal mechanisms underlying spasticity.

The Hoffmann reflex (H-reflex) is a compound muscle action potential elicited by electric stimulation of afferent Ia fibers in the mixed muscle nerve with subsequent recruitments of motor neurons through monosynaptic connections in the spinal cord.[3] The H-reflex is evoked by direct activation of Ia afferents and can reflect the excitability of the alpha motor neuron pool.[4] Several studies showed that the soleus H-reflex increased in spastic patients, but there are controversial reports.[5-9]

PAD is a frequency-related depression of the H-reflex and has been documented in both humans[3,10-16] and cats.[2,17] PAD of the reflex discharge has been described for stimulus intervals as long as 10 to 20 seconds.[18] PAD has been suggested to hold the synaptic efficacy of the Ia fiber at a relatively low level during voluntary movements and is of functional significance because it would keep the stretch reflex at a relatively low gain, and thus contributes to the prevention of clonus development.[14]

PAD can evaluate the integrity of adapted spinal circuitry. PAD has been shown to be impaired in spinal cord-lesioned animals[19,20] and in humans[3,11,21] with spasticity after spinal cord injury (SCI).

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Studies also showed that the decrease of PAD rather than the decrease of the amplitude of the H-reflex was related to the spasticity after SCI.[3,22]

The decrease of PAD after SCI is dependent on the time postinjury. In rats with spinal cord contusion[20] and spinal cord transection,[19] the magnitude of PAD was comparable with that of the control intact animals at acute phases postinjury, but at chronic phases of injury, the magnitude of PAD was significantly lower. Individuals with chronic SCI show less PAD compared with acute and nonimpaired individuals.[3,21]

Minimizing post-SCI complications, for example immobilization, might reverse these adaptations and consequently reduce spasticity. Decreased muscle tone has several clinical benefits, such as allowing patients to perform functional activities easier, wear orthosis, and perform home exercise programs. In order to remobilize paralyzed muscles after motor complete SCI, the therapeutic strategies are limited to passive exercise or electric stimulation. Electric stimulation has shown promising effects on the restoration of muscle properties.[23,24] However, no parallel restoration of the spinal circuitry function was found. Passive exercise used to be overlooked clinically because of the uncertainty of the therapeutic effect and its high demand for man power. The recent development of robot-assisted therapy makes the long-term passive exercise with high repetition possible, but the therapeutic effect on the restoration of spinal circuitry and clinical features needs to be clarified.

In transected animal studies, passive exercise can restore PAD. Four weeks of passive cycling attenuated and reduced the degree of atrophy in hind-limb muscles of spinal cord transected rats.[25,26] Skinner et al[19] conducted 3 months of 5 days per week passive cycling in acute spinal cord transected rats and showed the restoration of the PAD of the H-reflex to a level similar to the nonimpaired rats. Statistically significant levels of PAD were evident by 30 days of passive cycling exercise, although restoration of PAD could be seen starting 15 days postintervention.[27] A linear increase in PAD of the H-reflex was observed with a duration of 90 days of passive cycling implementation.[27] In summary, passive exercise is able to restore PAD in spinal cord transected animals in 4 weeks.

There are limited studies that document the effect of passive exercise on the restoration of spinal circuitry in humans with SCI. A case study reported that 1 patient with SCI who underwent 13 weeks of daily passive cycling showed gradual restoration of PAD with time. Subjective assessments indicated a significant reduction in spasticity.[28] However, the effect of passive cycling exercise has never been successfully reproduced in a controlled group study in order to set up clinical guidelines. A possible explanation is that passive cycling is a motion involving multiple joints of the lower extremities. The range of motion of the involved joints is difficult to control in cycling training. For example, excessive hip and knee movement might compensate the movement of a stiff ankle joint. The treatment effect is not easy to reproduce because of inconsistent range of joint movements across subjects.

List of abbreviations:

CPM	**continuous passive motion**
H-reflex	**Hoffmann reflex**
MAS	**Modified Ashworth Scale**
PAD	**postactivation depression**
SCI	**spinal cord injury**

The immediate decrease in reflex sensitivity after short-term intervention of continuous passive range of motion exercises of the ankle joint has been shown in both nonimpaired individuals and individuals with SCI.[29,30] There was also a decrease in the Modified Ashworth Scale (MAS) score, indicating a decrease of spasticity.[30] However, the long-term effect has to be established. The purpose of this study was to investigate if multiple sessions of continuous passive motion (CPM) of the ankle joint could restore the spinal circuit functions, PAD, after injury to the spinal cord once they had undergone adaptations. Because the single joint CPM will have better control of the range of motion for the joint to be trained than cycling motions, and animal studies showed promising restorations of PAD after 4 weeks of passive cycling training, the treatment duration was set at 4 weeks. One subject was overtrained for an additional 8 weeks to preliminarily explore the optimal duration of training. The results of this study could provide a new concept in clinical rehabilitation for individuals with chronic SCI and provide evidence for applications of the passive mode of robot-assisted therapy.

Methods

Fourteen subjects who had sustained a motor complete SCI for at least 6 months (chronic SCI) prior to testing were randomly allocated into either the 4-week ankle CPM training group or the control group (no training) (table 1). The randomization was according to a balanced prerandomized table. All subjects were recruited from the community. Subjects' medical conditions were stable. One subject allocated in the CPM training group volunteered to be overtrained for an additional 8 weeks after the end of the study in order to explore the dose response of training. All subjects had motor complete paralysis, which was corresponding to American Spinal Injury Association classification grade A or B,[31-33] at or above the T12 level. The sample size was determined by a pilot study, and the SD was estimated from previous research.[3]

All subjects gave their written informed consent in accordance with the Declaration of Helsinki. The study was approved by the institutional review board.

Experimental procedures

Prior to electrophysiologic testing, each subject underwent muscle tone evaluation of the ankle joint using the MAS in sitting position with knee flexion to 90°. This position was chosen to minimize the influence of gastrocnemius muscle tone. All clinical examinations were proceeded under identical conditions for each test by a licensed physical therapist who was blinded to group allocation. All subjects were seated with back support during electrophysiologic tests. The testing leg was determined in a randomized manner. The testing leg was secured with hook and loop straps on a forceplate system[30] with knee flexion 110°, and the slope of the forceplate was adjusted such that the ankle was supported in neutral position (0° dorsiflexion or plantarflexion).

The surface electromyographic recording electrode was positioned on the soleus muscle.[30,33-36] The recording electrode was positioned in parallel with the soleus muscle, approximately 2cm lateral to the midline of the distal calf and distal to the lateral head of the gastrocnemius, about two thirds the distance from the knee joint line to the lateral malleolus. A ground electrode was placed over the lateral malleolus. The M-waves and H-reflexes of the soleus were elicited by transcutaneous electric stimulation of the